

Ruptured Acute Appendicitis With Calcified Appendicolith Mimicking Acute Gastroenteritis

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Citation

M Akinkunmi, O Fadiran. *Ruptured Acute Appendicitis With Calcified Appendicolith Mimicking Acute Gastroenteritis*. The Internet Journal of Health. 2009 Volume 12 Number 1.

Abstract

A rare case of a man who presented with clinical features suggestive of acute gastroenteritis after a meal, and was subsequently diagnosed with ruptured acute appendicitis (AA) and calcified appendicolith on account of radiological findings, is reported. The diagnosis of AA can be difficult in the atypical case, and radiological imaging plays a vital role. The finding of calcified appendicolith in a patient presenting with acute abdomen should raise the index of suspicion of AA, especially in a developing country.

INTRODUCTION

Appendicitis is more common in developed than in developing countries and appendicoliths have an aetiologic role in the disease. The prevalence of appendicolith is higher in developed countries than in developing countries, and also higher in patients with than in those without appendicitis. This has been attributed to the low-fibre diets consumed in developed countries which lead to appendicolith formation¹. Calcified appendicoliths are found in 10% of patients with acute appendicitis, but they are seen more frequently in perforated appendicitis and in abscess formation². The clinical diagnosis of acute appendicitis (AA) is based primarily on patient history and on physical examination findings. The classic presentation of AA is seen in only 50% - 60% of patients, and the diagnosis may be missed or delayed in atypical presentations. The differential diagnosis of AA are abdominal pain of unknown cause, pelvic inflammatory disease and other gynaecologic disorders, mesenteric lymphadenitis, acute gastroenteritis and other gastrointestinal tract diseases, and urinary tract infection and obstruction³. This case is reported because firstly, it is uncommon for AA to mimic acute gastroenteritis in clinical presentation, secondly the calcified appendicolith was quite large, measuring 19 x 18mm, and thirdly to highlight the importance of radiological imaging in the diagnosis of acute abdominal pain.

CASE REPORT

AA is a 45 year old man who presented with the history of abdominal pain of sudden onset, diarrhoea, fever, headache,

and vomiting. His last meal (which he ate with other members of his family) was eight hours before presentation. No other member of the family had any of these symptoms. The past medical and surgical history and the drug history were not contributory. On examination, he was ill-looking, febrile (temperature of 37.6 degrees centigrade), and dehydrated. The pulse rate was 78/minute; regular and the blood pressure was 140/90 mmHg. The chest was clinically clear. The abdomen was full and mildly tender generally. The clinical impression was ?malaria, ?gastroenteritis secondary to food poisoning. He was commenced on intravenous (IV) fluids, IV hyoscine butylbromide, IV Maxolone, and intramuscular diclofenac. A plain abdominal radiograph was requested on account of increasing abdominal distension.

Abdominal ultrasonography revealed free extraluminal intraperitoneal fluid as evidenced by fluid in the hepatorenal recess. There was an aperistaltic, non-compressible, blind-ended, tubular structure in the right iliac fossa, measuring 13.9 mm in diameter, representing an inflamed appendix. There was periappendiceal inflammatory fluid and pericecal inflammation. The sonographic diagnosis was perforated AA with peritonitis, with perforated typhoid Ileitis as a differential diagnosis. Abdominal radiograph showed splaying of the properitoneal fat lines in keeping with abdominal distension, and a rounded, laminated, calcified mass (about 2 cm in diameter) in the right iliac fossa, representing a calcified appendicolith (Figure 1).

Abdominopelvic computed tomography (CT) scan showed a

rounded, high attenuation mass measuring 19 x 18 mm in diameter in the lumen of the distended appendix, indicating a calcified appendicolith (Figure 2). Minimal, free, extraluminal, intraperitoneal fluid was observed. The gall bladder, spleen, pancreas, and both kidneys were normal. Minimal, bilateral pleural effusion and mild consolidative changes of the basal segments of the lower lobes of both lungs were noted. The CT findings were conclusive of calcified appendicolith and perforated AA with peritonitis.

The stool, electrolytes and urea, and creatinine, and urinalysis were normal. Malarial parasites was negative; serum *Helicobacter pylori* was negative; and C-reactive protein (CRP) was non-reactive. Haemoglobin was normal. White blood cell count (WBC) was $9.52 \times 10^9/L$. (Neutrophils of 77.7%, lymphocytes and monocytes of 22.3%). Platelet count was $193 \times 10^9/L$. Liver function tests (LFT) and random blood glucose (4.8 mmol/L) were normal. He was commenced on IV ciprofloxacin and metronidazole, and had an emergency appendectomy with peritoneal toileting. The intraoperative findings were perforated gangrenous appendix distended with an appendicolith about 2 cm in diameter (Figure 3); feculent exudates in all intraperitoneal spaces measuring approximately 500 mls; and inflamed caecum and distal ileum. He made good clinical improvement and had delayed primary suturing done under local anaesthesia on the fifth day post-operation. He was discharged home on the eleventh day post-operation. He has remained well at every out-patient visit in the last two years post-surgery.

Histopathology confirmed a vermiform appendix about 7 cm long and 2.5 cm in diameter, with a rough and slightly hyperemic serosa. The cut section revealed lumen containing fecal matter and a calcified appendicolith measuring 1.5 x 1.6 x 1.4 cm. Microscopically, the histological section of the appendix showed hyperplasia of the mucosal lymphoid follicles and mucosal infiltrate of eosinophils and mononuclear cells extending into the submucosa. There were areas of necrosis of the entire wall thickening. There were fibrinopurulent inflammatory exudates covering the serosal layer. The pathologic diagnosis was acute necrotizing appendicitis with peritonitis.

Figure 1

Figure 1: Abdominal radiograph showing calcified, lamellated appendicolith (arrow).

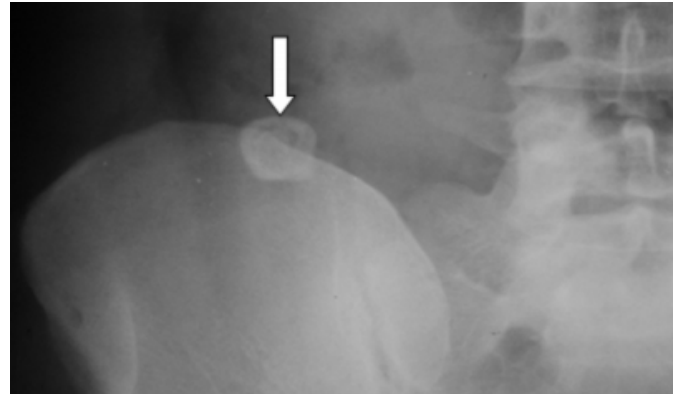


Figure 2

Figure 2: Nonenhanced abdominopelvic CT scan showing calcified appendicolith (arrow).

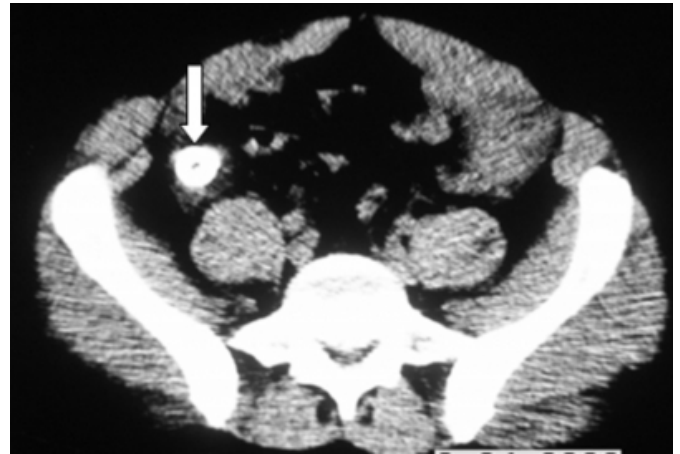


Figure 3

Figure 3: Photograph of the inflamed appendix removed at surgery.



DISCUSSION

The anatomy, pathophysiology, and clinical diagnosis of

AA, the role of CT and ultrasonography, and the effect of imaging on clinical outcomes, have been well described³. The surgical diagnosis of AA is customarily made on clinical grounds alone using history, physical examination, and white blood cell count in the typical presentation⁴. However in atypical presentations such as presented in this case, diagnostic imaging with CT and ultrasonography is usually required. The diagnosis of perforated AA was facilitated in the case presented by the radiological investigations. The clinical history and physical examination findings were not sufficient to clinch the diagnosis of AA. If anything, the diagnosis of acute gastroenteritis featured prominently in this case before the outcome of the ultrasound, plain abdominal x-ray, and CT examinations. In 1995, Ohmann et al assessed the value of different predictive scores in the diagnosis of AA; and found out that none of the Alvarado, Lindberg, Fenyo, and the Christian scores fulfilled any of the given criteria when applied to their database of acute abdominal pain and suspected AA⁵. The aim of managing AA is early surgical intervention to avert appendiceal perforation, which is associated with increased morbidity and mortality, compared with nonperforating appendicitis⁶. The ideal management is to subject the patient confirmed with the diagnosis of AA to early surgical intervention, while avoiding unnecessary appendectomy in the patient without AA. This decision may be very difficult in an atypical case. The ultrasound, plain abdominal x-ray and CT findings in this patient expedited surgical intervention. Opinion is divided on the relevance of the finding of appendicolith. The CT finding of appendicolith in a significant number of normal patients in a survey conducted by Huwart et al in 2006, made these investigators to suggest that appendicolith is not a specific sign for AA⁷. An appendicolith may be a marker of increased risk for appendicitis but is not an indication for appendectomy⁸. The calcified appendicolith was well shown on the plain abdominal radiograph in the index case. The definitive CT diagnosis of AA was made in this patient presented, on the finding of a calcified appendicolith in association with evidence of pericecal inflammation⁹. The identification of appendicolith on ultrasonography is highly associated with a positive diagnosis of AA; however failure to see an appendicolith on ultrasonography as exemplified in this index case is noncontributory³. The inflamed appendix with periappendiceal inflammation was well shown on ultrasonography in this patient, although it has been reported that the sensitivity and specificity for perforated AA are lower than those typically seen for nonperforated AA³. The

observations made by Jones et al regarding the higher prevalence of appendicolith and appendicitis in the developed world than in the developing countries, is instructive¹. The possibility of seeing a patient with appendicolith with clinical features suggestive of appendicitis is more likely in a developing country than in a developed country. The observations made by Forbes and Lloyd-Davies over 44 years ago on their evaluation of 1,800 appendices removed at surgery is still useful in the 21st century, and has stood the test of time. The high incidence of acute complications associated with a calculus urged them to recommend appendectomy when one is found incidentally on radiological examination of the abdomen¹⁰. This dictum is useful in communities where emergency medical services may not be readily available, as in many developing countries.

CONCLUSION

AA may masquerade as acute gastroenteritis in its clinical presentation. Whenever there is an atypical presentation of suspected AA, radiological imaging will be useful in further evaluation of such patients, as exemplified in the case presented. Calcified appendicolith may be seen in association with AA; and in a developing country this relationship appears to be more striking.

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